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ENVIRONMENTAL TOBACCO SMOKE AND PERIODONTITIS IN U.S. NON-SMOKERS

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A thesis submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Master of Science in Dental Hygiene Education in the Department of Dental Ecology, School of Dentistry.

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ABSTRACT

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Environmental Tobacco Smoke and Periodontitis in U.S. Non-smokers

(Under the direction of Dr. Anne Sanders)

The purpose of this study was to estimate periodontitis prevalence in non-smokers with detectable serum cotinine and investigate racial/ethnic and socioeconomic variation in ETS exposure. Data were from the 1999-2004 National Health and Nutrition Examination Survey (NHANES). Subjects were 3,137 lifetime non-smokers who had not used other forms of tobacco. ETS exposure was classified as negligible, moderate and high. Periodontitis was classified by the Centers for Disease Control and Prevention and American Academy of Periodontology case definition for moderate-severe disease. Survey estimation procedures were used to estimate prevalence and odds ratios (OR) in multivariable logistic regression models. ETS exposure was observed in 40.0% of subjects and 2.6% met the periodontitis case classification. ETS exposure was inversely associated with educational attainment and family income and was higher in non-Hispanic blacks than in whites. Exposure was higher among non-Hispanic blacks than whites and inversely associated with socioeconomic status. Higher ETS exposure was a risk indicator for periodontitis.

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I would like to sincerely thank my committee members: Dr. Anne Sanders, Dr. Leah Ranney, and Professor Rebecca Wilder for their diligent effort, and lengthy hours of editing, advice, and instruction. It has truly been a pleasure to have such high quality mentorship, and such well-placed encouragement through the process of becoming a scientific writer. I specifically thank Dr. Anne Sanders for the provision of this opportunity to work within such an inspired and significant study, and for the vast knowledge she possessed and demonstrated throughout the statistical analyses.

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Finally and greatly, I thank my husband, Joshua Sutton, for listening, for comforting, for encouraging, for prodding, and for kindling all my talents to grow. Truly, I stand taller knowing that he stands with me.

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LIST OF ABBREVIATIONS:

U.S.	**United States**
AAP	**American Academy of Periodontology**
CDC	**Centers for Disease Control and Prevention**
ETS	**Environmental Tobacco Smoke**
PD	**Pocket Depth**
CAL	**Clinical Attachment Level**
SES	**Socioeconomic Status**

INTRODUCTION

The American Academy of Periodontology (AAP) estimates prevalence of moderate to severe generalized periodontitis to be 30% or higher in the United States (U.S.) adult population, depending on the classification.[1] Periodintitis is a leading cause of tooth loss, tooth mobility and dental abscess; it is also positively associated with cardiovascular disease,[2] obesity,[3] Alzheimer's disease,[4] and diabetes mellitus.[5-12] The disease is characterized by chronic inflammation, loss of attachment, and bone loss. The condition is primarily caused by bacteria in dental plaque acting alone or in conjunction with systemic and genetic factors.[13] Other factors associated with the disease include psychological stress,[14] certain medications,[15, 16] genetics,[16] and tobacco use.[12, 17, 18] In fact, tobacco use is causally associated with periodontitis[12, 18-21] in a dose dependent relationship[22] and studies estimate the smoking attributable risk to be 20%.[1] Periodontal disease, although usually avoidable, impacts a large number of Americans and plays a role in other more serious and costly health problems.

Risk for disease increases with the number of cigarettes smoked, with notable differences observed in as few as 10 cigarettes per day.[22-25] Periodontal disease is 6-7 times as prevalent in the estimated 46 million adults in the U.S. who currently smoke.[26, 27] Smoking also makes the disease more virulent and difficult to treat. [21, 28, 29]

There is evidence of a relationship between periodontitis in non-smokers exposed to environmental tobacco smoke (ETS).[30, 31] Arbes, et al. observed that non-smokers with self-reported ETS exposure had 1.6 times the odds for periodontal disease compared to those not exposed.[30, 32, 33] The increased risk for periodontal disease occurs with the exposure to nicotine in cigarette smoke[30] which over-stimulates the host response in the oral cavity, complicating the already inflammatory nature of periodontal diseases.[34] In fact, the inflammatory response in salivary inflammatory markers is notable among those exposed to secondhand smoke.[35] However, to date measurements of ETS in the periodontal literature are limited to self-report and no objective biomarker of exposure has been examined.

ETS is unequally distributed between racial and ethnic groups. For physiological and behavioral reasons, non-Hispanic Blacks show higher concentrations of cotinine, with less exposure to cigarette smoke, than do non-Hispanic whites. Total and non-renal clearance of circulating cotinine is significantly lower in non-Hispanic blacks.[36] Furthermore, nicotine intake is 30% higher in African Americans, with a somewhat longer half-life for circulating cotinine.[36] The different absorption and manifestation of serum cotinine concentration in different races is supported by the prevalence periodontitis cases. According to Albandar and colleagues,[37] black and Mexican Americans display poorer periodontal health than whites with comparable income and educational attainment.[37] Signorello and colleagues[38] reported that "differences in cotinine levels among smokers suggest racial variation in exposure to/or metabolism of tobacco smoke constituents".[38]

Smoking and ETS are known hazards to health, including the oral cavity, and tooth attachment apparatus.[12] Together with racial and socioeconomic status, the differing levels of ETS exposure and different rates of metabolism for serum cotinine, provide a means and motivation to measure the rates of periodontal disease among the non-smoking population.[30] The aim of this study was to determine the prevalence of periodontitis in non-smokers with detectable serum cotinine, and to investigate the variation in ETS exposure among non-smokers classified according to racial and socio-economic characteristics.

REVIEW OF THE LITERATURE

The Surgeon General: Views on Tobacco

Since the 1950's, the harmful effects of smoking have been hypothesized and researched. Active cigarette smoking was implicated formally in the cause of disease in 1964 when the Surgeon General and advisory committee determined that irrefutable evidence existed for a causal association between smoking and lung disease.[39] Since that time, more than twenty reports published by the Surgeon General have spoken to the hazards of smoking. In 1972, involuntary smoking (second hand smoke) was implicated as hazardous by the Surgeon General.[40] Involuntary smoking continued to surface in 1986,[41] 1994,[42] 2004[12] and 2006.[43] These reports gave evidence to a changing social perception of smoking; initially, smoking was known to be dangerous first hand, but was later found to be dangerous first and second hand, culminating with decreased social acceptance. In impetus, the 2004 report comprised 960 pages of health consequences of smoking, and added periodontal disease to the already lengthy list of diseases known to be caused by smoking.[12] The 2006 report drew major conclusions about tobacco smoke, with Surgeon General Richard Carmona issuing the following statement: "there is no risk-free level of exposure to secondhand smoke".[43]

Examples of Government Intervention designed to improve population Health in the U.S.

Today, smoking is considered by the United States Centers for Disease Control and Prevention (CDC) to be the leading cause of preventable death, with 443,000 of those deaths in the United States alone.[44] Of those nearly half a million deaths, roughly 50,000 are due to indirect tobacco use, or second hand smoke exposure.[44] Per the 2006 report, there is evidence that first and second hand smoke, even at low levels of exposure cause disease.[43]

The United States has a commissioned corps of health responders known as the U.S. Public Health Service. The mission statement for the organization embodies the value placed on health and wellbeing in America: "the mission of the U.S. Public Health Service Commissioned

Corps is to protect, promote, and advance the health and safety of our Nation".[45] Several examples of intervention in health care and prevention exist and are discussed in the following paragraphs.

One of the most noteworthy American public health interventions was the fluoridation of drinking water in the late 20th century.[46] In 1901, a dentist's interest in mottled tooth enamel led to further investigation of its etiology. Using years of study, and epidemiological evaluation of the trend linking fluoride intake to caries prevention, this discovery and its clinical relevance led to the implementation of meaningful change. Fluoride is responsible for a 68% decrease in the number of decayed, missing, and filled teeth in 12 year olds from the 1960's to the 1990's.[46-48]

According to Cox, et al., influenza is the most frequent cause of acute respiratory illness requiring medical intervention.[49] Annually, the CDC's Advisory Committee on Immunization Practices urges those in the American population who are medically compromised or otherwise susceptible to influenza to receive an updated attenuated vaccine for the prevention of influenza. This comes in response to decreased viral influenza infection using vaccination-- in fact, since 1968; accurate vaccination is credited with the prevention of influenza pandemic, with only one exception.[50]

Additionally, for admission and registration into public school and childcare environments, vaccination is required. The actual vaccinations required vary by state, with most requiring Varicella, Hepatitis, Measles, Mumps and Rubella, as well as Diphtheria, Tetanus and Pertusus before the completion of grade 12.[51] This intercession on the part of the US government is to prevent disease based on clinical evidence that vaccination works: in the case of Varicella vaccination, the one-dose vaccine was found to be 98.1% effective[51] and is therefore required by most states for children.

Deficiency in folic acid has led to many different birth defects including spina bifida and anencephaly, stemming from Neural Tube Defects.[52] Evidence such as this led to the passage of the Birth Defects Prevention Act of 1998.[53] Under this bill, law 105-168 authorizes the Centers for disease control to collect information on birth defects, perform research on observed birth defects epidemiologically, and inform and educate the public about birth defects.[53] The observation of Neural Tube Defects, as well as the link to their prevention using folic acid has led to daily guidelines for women with suggested daily folic acid consumption, and food fortification in cereals and flour.[54-56] In an article published in the Food and Nutrition Bulletin in 2010, the CDC stated that mandatory fortification of flour with folic acid in the U.S. has been one of the most successful interventions in public health for reducing the prevalence of Neural Tube Defect-scarred pregnancies.[57]

Interventions exist in the United States for the betterment of public health ranging from water fluoridation to vaccination, to dietary fortification of foods, however an appropriate level of protection is still debated in cigarette smoking and smoke exposure. Immediately after the 1964 report on smoking and health by the Surgeon General, congress passed the Cigarette Labeling and Advertising Act, mandating that every pack of cigarettes include a warning label: "Caution: cigarettes are hazardous to your health".[58] Further, in 1984 Congress passed the Comprehensive Smoking Education Act which required four rotating health warning labels on packets of cigarettes:[58]

1. "Smoking causes lung cancer, heart disease and may complicate pregnancy."
2. "Quitting smoking now greatly reduces serious risks to your health."
3. "Smoking by pregnant women may result in fetal injury, premature birth, and low birth weight."
4. "Cigarette smoke contains carbon monoxide."

This act also encouraged the reporting of relationships between smoking and health biennially to Congress, as well as the provision of a confidential list of ingredients added to cigarettes manufactured in or imported into the United States.[58] Throughout the 80's and 90's, smoking was banned on short flights (< 2 hours), then longer ones (<6 hours), in federally funded children's services, and in 'Women, Infants and Children (WIC) clinics'.[58] In 2009, the Food and Drug Administration was granted authority over the regulation of tobacco products, and states continue to change and increase bans on indoor and public smoking regulation.[58] Most interventions historically focused on the cigarettes themselves; on their labeling, sales and consumption. But increasingly, the United States has become interested in and active toward the protection of non-smokers from cigarette smoke's hazardous effects.[58]

Health Hazards of Passive Smoking

Of each lit cigarette, 15% of the smoke generated is inhaled by the smoker as mainstream (firsthand) smoke, while 85% is either exhaled by the smoker, or expelled from the burning tip of the cigarette.[59] In addition to firsthand smoke, secondhand smoke is implicated in diseases noxious to the American public, such as exacerbation of asthma, bronchitis, pneumonia, Chronic Obstructive Pulmonary Disease (COPD), sudden infant death syndrome (SIDS), middle ear disease in children and cancer.[43, 60, 61] Increased exposure to second hand smoke also causes sub-disease level irritation of the eyes, and slows lung growth in exposed children.[43] Also, mothers exposed to ETS are prone to deliver babies with reduced birth weight.[43]

As of 2007, environmental tobacco smoke (ETS) exposure was one of the top three causes of preventable death in the United States.[62] Cigarette smoke is known to contain harmful substances, such as carbon monoxide, formaldehyde, cyanide, ammonia and nicotine,[43] among others. It also contains greater than 50[43] known carcinogens, such that cigarette smoke is categorized a Class A carcinogen by the Environmental Protection Agency (EPA).[63] As such, it costs the United States roughly $10 million annually in non-smoker health care costs to treat smoking related illness.[44]

In addition to life threatening systemic diseases, ETS affects oral health. The evidence surrounding active smoking, and periodontitis is enough to infer a causal relationship; therefore, active smoking causes periodontitis.[12]

Periodontitis

The CDC in conjunction with the American Academy of Periodontology (AAP), have determined a case definition of periodontitis for population-level research. This case definition describes periodontitis as a collection of periodontal pockets or clinical attachment loss, intraorally, at a level >4mm, in two or more non-adjacent sites.[64] The Glossary of Terms released by the AAP defines periodontitis as an inflammation of the supporting tissues of teeth that is usually progressive and destructive leading to loss of bone and connective tissue.[65] This inflammation can extend from the gingiva into adjacent bone and attachment apparatus.[65] It is caused by a biofilm of plaque consisting of specific and numerous bacteria, acting alone or in concert with complicating systemic factors.[66]

National Health and Nutrition Examination Survey data released between 1999 and 2004 yield a representative sample of the United States population and a 3.6% estimate of national prevalence of periodontal disease.[67] However, Dr. Eke (lead epidemiologist for the CDC) reported in 2010 that the value likely underestimates the actual prevalence of disease due to the limited nature of periodontal examination in NHANES evaluation.[68]

Environmental Tobacco Smoke and Periodontitis

Within the estimated 3o% of Americans with periodontal disease,[1] current smokers exhibit higher rates of disease.[24] When compared with non-smokers, smokers are 9% more likely to have disease.[64, 69] Active smoking alters microbial and host response factors in periodontitis, and has been implicated in bone loss, such as osteoporosis.[34] In respect to microbes, preliminary findings by Teughels et al. indicate that individual periopathogens' (*A. Actinomycetemcomitans*, and *P. Gingivalis*) colonization of tissues could be impacted by nicotine in a species-specific manner.[70] Passive smoking, like active smoking, impacts the immune response, namely polymorphonuclear leukocyte (PMN) function such as phagocytosis, chemotaxis and oxidative

burst.[71] As reported by Numabe et al. in 1998, phagocytic activities of PMN's intensify after smoking and passive smoking.[71] Additionally, the results suggested that certain substances in smoke overstimulate the host response in the oral cavity[71] making the exposed more likely to experience attachment and toothloss.[72]

According to Arbes et al., periodontal disease was 1.6 times as likely among non-smokers with self-reported exposure to ETS[73] than those not exposed. In 2006, Nishida et al. attempted to determine the mechanism of passive smoking when considering the development of periodontitis, and evaluated the manifestation of passive smoke exposure of salivary markers related to periodontitis.[74] The result was an elevated concentration of inflammatory makers Interleukin - 1β, Albumin and Aspartate Aminotransferase (AST), in those exposed to passive smoke.[74] Further, Nishida et al. determined that the evidence regarding longitudinal influence of involuntary smoking on periodontitis progression was insufficient. In response, they conducted a study of Japanese employees during annual health checkups from 2003 to 2005.[35] Their study concluded that the risk for periodontitis progression was greater for those exposed to involuntary smoke, along with increased inflammatory response, intraorally.[35] Further, Shizukuishi et al. added to Nishida's work, concluding that by stimulating inflammatory responses in periodontal tissue, "passive smoking exposure may be a risk factor of periodontal disease".[34]

Socio-Economic Status, Health and Behavior

In 2010, Nettle of the U.K. presented a model of why socioeconomic gradients in health behavior might exist.[75] His model predicted that unavoidable exposure surrounding an impoverished life could lead to "disinvestment in health behavior".[75] His model predicted a greater inequality in health outcome than that which originally existed in life conditions.[75] In Wisconsin, a 2001 study by Malmstadt et al. concluded that study participants with less than a high school education displayed smoking prevalence 28% higher than those with a college degree or more.[76] Further, another study in Wisconsin indicated that people with incomes between $10,000 and $15,000 dollars per year were more than twice as likely to smoke as those with higher incomes.[77] Elsewhere, of inner city African-American, Latino and White study participants, Siegel et al. determined that ethnicity and education predicted smoking, with White and African-Americans 3.2 and 1.7 times as likely to smoke as Latinos, respectively.[78]

Socioeconomic status is also recognized as a gradient affecting disease prevalence. In the European Heart Journal, Mackenback et al. wrote that "in all countries, mortality from cardiovascular disease is higher among persons with lower occupational class, or lower education level".[79] In 2010, the Public Health Report by the CDC sampled participants representative of Americans; findings included income and education gradients in diabetes-related mortality.[80]

Additionally, Dray-Spira et al. agreed that the risk of mortality differs substantially according to education level among individuals with diabetes in the U.S.[81] Overall, the health of people in the United States is heavily affected by socioeconomic and ethnic/racial factors. Those with the lowest income, and the least education are consistently less healthy, with health gradually increasing with income and education.[82] For example, among insulin dependent diabetics in the United States, those with low income and Hispanic ethnicity were less likely to regularly monitor their blood glucose levels.[83] Income and education gradients present in general health, are also present in oral health with poorer clinical and perceived health resting firmly in lower income and education levels.[84] According to Drury et al., caries, gingivitis, and loss of periodontal attachment were observed in American participants with lower socioeconomic status as defined by income and educational attainment.[85] Finally, a 2008 review of National Health and Nutrition Examination Survey (NHANES) data released between 1999 and 2004 indicated pervasive inequalities in periodontitis prevalence associated with race/ethnicity, education and income,[67] specifically, African American periodontitis prevalence was 7.2%, as opposed to 4.4% of Mexican Americans, and 3% for White Americans.[67] Of the participants, periodontitis was found most among Black participants (CI 95%, 1.94), those with less than a high school education (95% CI, 2.06) and those with low income (95% CI, 1.89).[67]

Cotinine and Nicotine Exposure

The permeation of cigarette smoke can be difficult to measure and determine. Self reported exposure could be somewhat subjective. A biological measure exists. Using an effective, quantifiable biomarker, accuracy can be achieved in measuring environmental tobacco smoke exposure.[86] According to the National Research Council, there are criteria that must be met for effective biomarkers, such as unique isolation from only tobacco smoke, ease of detection, emission at similar rates for a variety of products, and a constant representation of ETS.[86] Cotinine is such a valid marker in serum for ETS exposure.[86] While this value is a constant and quantifiable average, with predictable correlations to exposure, it does differ somewhat by individual physiology, found in differing characteristics such as sex, race, and age.[86] With alarming sensitivity, cotinine has been used to confirm ETS related illness to exposed non-smoking children;[87]; cotinine reflects exposure, especially under sustained exposure conditions.[87] According to the Surgeon General, currently, cotinine (the primary proximate metabolite of nicotine) "remains the biomarker of choice for assessing secondhand smoke exposure".[88]

Purpose

It is well documented that exposure to first- or secondhand smoke[24, 41, 89] increases disease risk; it is also understood that the most accurate measurement for exposure to second-hand smoke

is nicotine's metabolite: cotinine. What remains to be documented is whether tobacco smoke exposure as measured by serum cotinine significantly is associated with periodontitis in non-smokers, and if so, whether a notable socio-demographic gradient exists in exposure or disease.

INTRODUCTION AND REVIEW OF THE LITERATURE

The American Academy of Periodontology (AAP) estimates that 30% or more of the United States (U.S.) population suffers from moderate to severe generalized periodontitis, depending on the classification.[1]Periodintitis is a leading cause of tooth loss, tooth mobility and dental abscess; it is also positively associated with cardiovascular disease,[2] obesity,[3] Alzheimer's disease,[4] and diabetes mellitus.[5-12]The disease is characterized by chronic inflammation, loss of attachment, and bone loss. The condition is primarily caused by bacteria in dental plaque acting alone or in conjunction with systemic and genetic factors.[13] Other factors associated with the disease include psychological stress,[14] certain medications,[15, 16] genetics,[16] and tobacco use.[12, 17, 18] In fact, tobacco use is causally associated with periodontitis[12, 18-21] in a dose dependent relationship[22] and studies estimate the smoking attributable risk to be 20%.[1] Periodontal disease, although usually avoidable, impacts a large number of Americans and plays a role in other more serious and costly health problems.

Risk for disease increases with the number of cigarettes smoked, with notable differences observed in as few as 10 cigarettes per day.[22-25] Periodontal disease is 6-7 times as prevalent in the estimated 46 million adults in the U.S. who currently smoke.[26, 27] Smoking also makes the disease more virulent and difficult to treat. [21, 28, 29]

There is evidence of a relationship between periodontitis in non-smokers exposed to environmental tobacco smoke (ETS).[30, 31] Arbes, et al. observed that non-smokers with self-reported ETS exposure are at 1.6 times the odds for periodontal disease compared to those not exposed;[30] confirming that ETS is independently associated with periodontitis.[32, 33] The increased risk for periodontal disease occurs with the exposure to nicotine in cigarette smoke[30] which over-stimulates the host response in the oral cavity, complicating the already inflammatory nature of periodontal diseases.[34] In fact, the inflammatory response in salivary inflammatory markers is notable among those exposed to secondhand smoke.[35] However, to date measurements of ETS in

the periodontal literature are limited to self-report and no objective biomarker of exposure has been examined.

ETS is unequally distributed between racial and ethnic groups. For reasons both physiologically and behaviorally based, non-Hispanic Blacks manifest higher concentrations of cotinine, with less exposure to cigarette smoke, than do non-Hispanic whites. Total and non-renal clearance of circulating cotinine is significantly reduced in non-Hispanic blacks ($p=.009$).[36] Furthermore, nicotine intake is 30% higher in African Americans, with a somewhat longer half-life for circulating cotinine.[36] The different absorption and manifestation of serum cotinine concentration in different races is supported by the prevalence periodontitis cases. According to Albandar and colleagues,[37] black and Mexican Americans display poorer periodontal health than whites with comparable income and educational attainment.[37] Signorello and colleagues[38] reported that "differences in cotinine levels among smokers suggest racial variation in exposure to/or metabolism of tobacco smoke constituents".[38]

Smoking and ETS are known hazards to health, including the oral cavity, and tooth attachment apparatus.[12] Together with racial and socioeconomic status, the differing levels of ETS exposure and different rates of metabolism for serum cotinine, provide a means and motivation to measure the rates of periodontal disease among the non-smoking population.[30] The aim of this study was to determine the prevalence of periodontitis in non-smokers with detectable serum cotinine, and to investigate the variation in ETS exposure among non-smokers classified according to racial and socio-economic characteristics.

MATERIALS AND METHODS:

Study and Sampling Designs

This cross-sectional study is nested within a larger study designed to examine the relationship of a state's cigarette excise tax on cigarette sales and levels of ETS. Data were obtained from the National Health and Nutrition Examination Survey (NHANES) release dates 1999-2000, 2001-2002, and 2003-2004. The NHANES is an ongoing representative survey of the health and nutrition status of the civilian, non-institutionalized U.S. population, conducted by the National Center for Health Statistics (NCHS).[90]

The NHANES uses a complex cross-sectional survey design to sample participants 2 months of age and older.[30] Because NHANES typically samples 15 primary sampling units per survey, the current study combined three survey releases to maximize the number of sampled states.

Data Collection

Data collection consisted of a household interview, and a medical examination including a dental component, conducted in the Mobile Examination Centre (MEC). The household interview included questions pertaining to socioeconomic characteristics, medical/dental history, and health behaviors, such as smoking. During the physical examination, blood was collected by venepuncture to allow for serum cotinine measurement in participants over 3 years of age.[91] no we didn't obtain consent. This is secondary data analysis.

Participants

In the combined 1999-2004 NHANES data, 9,932 adults aged 20 years or older received a periodontal assessment. Those who reported having smoked at least 100 cigarettes in their lifetime (n=4,553) were precluded from analysis. Also precluded were 13 adults with undisclosed smoking status, along with individuals with a history of tobacco use through pipe, cigar, snuff or chewing tobacco (n=456). Examination of serum cotinine identified participants whose sex- or race/ethnicity-specific concentrations exceeded thresholds for non-smokers

(n=437) and these were likewise ineligible. Finally, adults having lived in the U.S. fewer than 10 years were precluded (n=1,336) since ETS exposure in these individuals could not be related to the state-level excise as this study is a nested within a greater investigation of tobacco excise tax and its relationship to periodontitis. Hence this analysis was limited to 3,137 U.S. lifetime non-smokers.

Dependent Variable

An assessment of periodontal tissues was conducted by a licensed dentist during the NHANES oral examination. Examination measured bleeding on probing and periodontal pocket depth for two randomly assigned quadrants: one maxillary and one mandibular. Probing was done using a National Institute of Dental Research (NIDR) probe.

The assessment included permanent fully erupted teeth, excluding root tips, partially erupted teeth, and third molars. Measurements used were taken from the mesial and mid-buccal aspects of the teeth from distal to mesial, beginning with the distal-most tooth, moving toward the midline. Over the six-year survey period, periodontal measurement techniques differed. For release dates 1999-2000, periodontal measurements were taken at two sites on each assessed tooth: midbuccal and mesiobuccal. For release dates 2001-2002, and 2003-2004, measurements were collected from the midbuccal, mesiobuccal and distobuccal sites of teeth. For consistency during analysis, the mesiobuccal numbers were analyzed for the entire survey period, as interproximal sites pertain directly to the case definition used.

Periodontal cases were defined using a case classification developed by the CDC and the AAP. [64] The AAP defines periodontal disease as "two or more interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or two or more interproximal sites with probing depth ≥5 mm, not on the same tooth".[64]

Key Exposure Variable

Questions about smoking history and use of tobacco products were presented in the household Environmental tobacco smoke exposure was measured using serum cotinine measurements collected during the medical examination. Exposure was defined as serum cotinine measurements ≥0.05 ng/mL as this is the NHANES laboratory-limit for detection. The use of the biomarker cotinine was indicated due to its ability to reflect nicotine exposure over days and its specificity to nicotine;[86] evaluating only recent cigarette smoke exposure as opposed to all environmental inhaled substances.[87]

Independent Variables

Along with tobacco smoke exposure, the characteristics age, sex, educational attainment, annual family income and ethnicity were considered independent variables. These characteristics were identified during the household interview questionnaire

RESULTS

In this non-smoking subset of the general U.S. population, males and all individuals with low levels of education and family income were under-represented. According to serum cotinine concentrations, 40.5% of participants were exposed to ETS (Table 1). Greater proportions of males than females were exposed and adults aged 20-49 years were more likely to be exposed than were their older counterparts ($p<0.001$). Most pronounced differences in ETS exposure were found between racial groups. Two-thirds of African Americans were exposed compared with approximately one third of Non-Hispanic whites ($p<0.001$). Even within this advantaged subset of the U.S. population, inverse socioeconomic gradients were observed in levels of ETS exposure (Table 1).

The CDC/AAP case classification for moderate or severe periodontitis was met by 2.6% of participants (n=82) (Table 2). Of note, serum cotinine concentration was not significantly associated with periodontitis in unadjusted analysis. In addition, the associations of periodontitis with sex and race/ethnicity were statistically non-significant, while age and socioeconomic status were strongly associated with the disease. Odds of periodontitis were elevated nine-fold in adults with incomplete high school education relative to those with at least some college education (OR =9.1; 95% CI: 5.2, 15.9). In the multivariable model (Table 3), that adjusted for potential confounding of age and other factors, odds of periodontitis was 89% higher in adults with cotinine concentration $\geq$1.5ng/mL compared to those with negligible concentrations. The predicted probability of meeting the periodontitis case classification increased monotonically with increasing levels of serum cotinine concentration (Figure 1).

DISCUSSION

This study sought to evaluate the relationship between environmental tobacco smoke and periodontitis in non-smokers using an objective biomarker. The primary finding was that periodontitis in non-smokers is positively influenced by environmental exposure to environmental tobacco smoke. This stands in agreement with similar previous studies: Arbes et. al[73] found a relationship between self-reported smoke exposure and periodontitis in non-smokers,[73] while a study comparing salivary cotinine to periodontitis also found an increase in salivary markers related to periodontitis with exposure.[35, 74] NHANES data provided a representative sample of the American population, as well as a large sample size for analysis. Moreover, it allowed for analysis of tobacco use in addition to cigarettes alone. Specifically, it allowed for the study of participants controlled for cigar, pipe, and smokeless tobacco use. Both the medical history questionnaire in the NHANES protocol, and the serum concentration tests for serum cotinine added to reporting accuracy.

This study evaluated data from 1999-2004. Since that time regulations controlling exposure of ETS to non-smokers have changed. For example, in 2009, the Family Smoking Prevention and Tobacco Control Act was passed granting the Food and Drug Administration (FDA) the authority to regulate tobacco products.[58] Among the states, North Carolina recently passed tobacco control legislation to ban cigarette smoking in restaurants as of January 20, 2010.[92] Of the 50 states in America, 50% of the U.S. population was protected by some combination of Clean Air policies as of 2008.[93] Recent tobacco control acts undoubtedly changed who is exposed to cigarette smoke and at what rate.

Another limitation of the data is that NHANES protocol allows for half-mouth data collection, with limited periodontal reading sites per tooth during the periodontal assessment. However, officials at the CDC concede that this abbreviated assessment protocol underreports periodontitis prevalence.[68] The periodontal assessment protocol changed throughout the five years of data collection reported in this study; therefore, collected data were reduced to the two common sites per tooth. Additionally, NHANES reports that trained dentists performed the

periodontal assessments, but no kappa score is reported for intra-rater reliability. The questionnaires and testing methods do not identify in which locale the participants were exposed to second hand smoke. For this reason, it is difficult to know which improvements should be made to tobacco control policy.

Unexpectedly, the threshold of harmful exposure differed between racial groups. For example, from the same exposure, non-Hispanic blacks absorb 30% more cotinine than do non-Hispanic whites.[36] Greater absorption of ETS may explain why non-Hispanic blacks were more likely to have periodontitis than non-Hispanic whites. Also unexpected was the finding that younger adults were less likely to have periodontitis, while being more exposed to cotinine, however, age is an associated risk factor for periodontitis[16, 94] due to lifetime disease, and CAL accumulation. The increased exposure in younger adults could be due to lifestyle differences,[36] exposure environments, and personal oral hygiene habits.

Studies have previously linked cigarette smoking to race,[76] as well as social gradients in periodontitis.[37, 75] Therefore, the strong gradient found between income level and cotinine exposure, as well as the one found between education level and exposure[77, 95] were expected. In general, the study methods used here could be implemented in any other nationally representative examination. This study echoes the finding of income, education and race gradients between exposure and disease, It also confirms that tobacco control bans are beneficial[96, 97] and should increase in the future as they decrease public smoking, and the permeability of environmental tobacco smoke. Future research could evaluate in what specific ways public smoking bans are beneficial to non-smoking, at-risk populations.

Currently, psychological tools[98] and assessment instruments[99] are used to encourage meaningful and motivated behavior change in patients, as well as increase provider confidence in providing cessation techniques.[99] This study has strong and timely implications for dental hygiene practice. An update on clinical practice guidelines in the way of smoking cessation counseling estimated a two fold increase in smoking cessation counseling since the early 1990's,[100] as well as a steadily decreasing rate of smokers.[100] Multiple controlled trials report efficacy in tobacco cessation counseling, indicating that moments shared by patients and providers in dental care settings are teachable moments,[101] and that patients listen and are encouraged by the focus on individualized oral health, for instance, patients are more likely to approach tobacco behavior change in response to existing oral complaints such as tooth color, or oral malodor that can be associated with smoking.[102] For that reason, as well as the documented link between cigarette smoke and systemic disease,[60, 62, 89] this study is crucial.

Dental hygienists are in a powerful position to affect future behaviors of patients by utilizing those teachable moments to relate to patients and identify those at risk. Research demonstrates that flexibility in tobacco education curriculum encourages incorporation of tobacco education in dental hygiene programs.[103] In an ever expanding body of research, the curriculum should expand to include the most recent evidence: that ETS affects the periodontal health of even non-smoking patients. This, along with continued research could further strengthen the education provided to patients as well as the confidence with which it is delivered.[104]

The strong relationship found between serum cotinine and increased odds of periodontitis provides evidence that mere smoking cessation counseling is not enough: education about dangers of cigarette smoke should also express the dangers of passive smoke exposure. This finding holds importance for healthcare providers in a position to advise and educate patients. Since a large percentage of those unwillingly exposed to second hand smoke are children,[105] an effort to inform parents though public health initiatives and stronger tobacco control policies for homes and cars would be valuable.

In the future, similar studies with more recent release dates are needed to compare the differences in exposure to non-smokers as tobacco control policy increases. Sub-grouped participants in areas of high tobacco control, moderate, and low areas of tobacco control would further identify the benefit of reducing exposure, particularly in areas with disadvantaged populations. Due to the strong socioeconomic gradients, studies of the knowledge and opinions about passive smoke of "at-risk" groups could illuminate shortcomings in education to protect those most at risk of exposure and help to advance tobacco control policies.

CONCLUSION

Cigarette smoke is harmful to periodontal health, irrespective of whether exposure is voluntary or not. This study was the first to quantify the presence and concentration of environmental tobacco smoke (ETS) exposure using serum cotinine as a biomarker for exposure in a representative population of U.S. non-smokers.

Striking racial and socioeconomic gradients between ETS exposure and periodontitis were observed. Compared with whites, greater proportions of African Americans were exposed to ETS and had moderate or severe levels of periodontitis. Lower levels of education and income were associated with higher ETS exposure and disease. In all, forty percent of non-smokers were exposed to ETS during 1999-2004, and exposure was significantly associated with two-fold higher odds of moderate or severe periodontitis.

Table 1. Selected characteristics of the dentate non-smoking population aged 20 years or older, resident in the United States for ≥10 years, and the percentage exposed to environment tobacco smoke (ETS) (N=3137), NHANES 1999-2004

Characteristic	Unweighted N and weighted %	Exposure to ETS (%) [a]	95% CI	P value
All	3,137 (100.0)	40.5	35.9, 45.2	
Sex				
Male	1,090 (36.9)	46.4	40.3, 52.6	<0.001
Female	2,047 (63.1)	37.0	32.7, 41.6	
Age group (years)				
20-49 years	2,003 (69.7)	43.9	38.9, 48.9	<0.001
50-85 years	1,134 (30.3)	32.6	27.7, 38.0	
Race/ethnicity				
Non-Hispanic White	1,858 (79.2)	36.2	31.1, 41.7	<0.001
Non-Hispanic Black	718 (12.4)	65.7	60.0, 71.1	
Hispanic	522 (6.9)	41.1	33.1, 49.7	
Other	39 (1.5)	51.3	31.4, 70.7	
Educational attainment				
Less than high school	513 (9.8)	58.4	51.0, 65.5	<0.001
High school graduate or equivalent	725 (22.7)	50.7	44.6, 56.7	
Some college or more education	1,898 (67.5)	34.4	29.4, 39.8	
Missing	1			
Annual family income				
<$25,000	930 (24.5)	54.2	47.5, 60.7	<0.001
$25,000-<$75,000	1,352 (44.4)	40.7	34.8, 46.8	
≥$75,000	756 (31.2)	29.1	23.7, 35.2	
Missing	99			

[a] Environmental tobacco smoke exposure was determined by sex- and race-specific thresholds of serum cotinine above the laboratory detection limit for 1999-2000 NHANES of 0.05ng/mL

[b] All estimates are weighted data, except the number of study participants, which is reported unweighted.

Table 2. Mean (95% CI) serum cotinine level (ng/mL), prevalence of periodontitis (95% CI) and odds ratios for periodontitis (OR) (95% CI) according to sociodemographic characteristics of study participants (n= 3,137), NHANES 1999-2004

Characteristic	Serum cotinine (ng/mL) mean (95% CI)	P-value	Periodontitis [a] prevalence (95% C.I.)	P-value	OR periodontitis (95% CI)
All	0.20 (0.18, 0.23)		2.61 (2.08, 3.26)		–
Sex					
Male	0.25 (0.21, 0.30)	<0.001	2.16 (1.46, 3.17)	0.302	1.34 (0.76, 2.36)
Female	0.17 (0.15, 0.20)		2.87 (2.09, 3.92)		Ref
Age group					
20-49 years	0.23 (0.20, 0.27)	<0.001	0.49 (0.32, 0.75)	<0.001	Ref
50-85 years	0.13 (0.10, 0.16)		7.46 (5.98, 9.28)		16.27 (10.49, 25.23)
Race/ethnicity					
Non-Hispanic White	0.16 (0.14, 0.19)	<0.001	2.33 (1.78, 3.04)	0.146	Ref
Non-Hispanic Black	0.50 (0.40, 0.59)		4.07 (2.84, 5.80)		1.78 (1.10, 2.88)
Hispanic	0.11 (0.08, 0.13)		2.70 (1.56, 4.63)		1.16 (0.64, 2.12)
Other	0.18 (0.06, 0.30)		4.79 (0.90, 21.77)		2.11 (0.42, 10.65)
Educational attainment[b]					
< High school	0.44 (0.33, 0.54)	<0.001	9.48 (6.96, 12.80)	<0.001	9.07 (5.16, 15.94)
High school or equivalent	0.26 (0.20, 0.32)		4.00 (2.60, 6.09)		3.60 (1.95, 6.65)
≥ Some college	0.15 (0.12, 0.18)		1.14 (0.75, 1.72)		Ref
Annual family income[b]					
<$25,000	0.36 (0.28, 0.43)	<0.001	5.24 (3.77, 7.25)	<0.001	6.27 (2.45, 16.04)
$25,000-<$75,000	0.19 (0.15, 0.22)		2.32 (1.61, 3.33)		2.69 (1.07, 6.72)
≥$75,000	0.10 (0.07, 0.13)		0.88 (0.38, 2.00)		Ref
Serum cotinine concentration[c]					
<0.05 ng/mL	0.02 (0.02, 0.03)	<0.001	2.33 (1.66, 3.27)	0.509	Ref
0.05 - <0.15 ng/mL	0.09 (0.08, 0.09)		3.06 (1.94, 4.80)		1.32 (0.73, 2.40)
≥1.5 ng/mL	0.82 (0.75, 0.90)		2.97 (1.96, 4.48)		1.28 (0.74, 2.22)

[a] CDC/AAP case classification for moderate or severe periodontitis defined as ≥2 interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or ≥2interproximal sites with probing depth ≥5 mm, not on the same tooth

[b] Fewer than 3,137 subjects were analyzed because of missing data

[c] The laboratory detection limit fort 1999-2000 NHANES (0.05) was applied for all years (1999-2004)

Table 3. Multivariable analysis modeling odds ratio (OR) and 95% confidence interval (CI) for moderate or severe periodontitis [(a)] in dentate non-smoking United States adults aged ≥20 years [(b)] (n= 2,998), NHANES 1999-2004

Characteristic	OR (95% CI)
Sex	
Male	1.17 (0.65, 2.12)
Female	Ref
Age in years	1.08 (1.06, 1.10)
Race/ethnicity [(c)]	
Non-Hispanic white	Ref
Non-Hispanic black	2.52 (1.35, 4.71)
Hispanic	1.70 (0.81, 3.58)
Educational attainment	
Less than high school education	2.74 (1.45, 5.21)
High school graduate or equivalent	1.82 (0.89, 3.71)
Some college or more education	Ref
Annual family income	
<$25,000	1.79 (0.68, 4.70)
$25,000-<$75,000	1.42 (0.57, 3.56)
≥$75,000	Ref
Serum cotinine concentration	
<0.05 ng/mL	Ref
0.05 - <0.15 ng/mL	1.16 (0.62, 2.18)
≥1.5 ng/mL	1.89 (1.08, 3.31)

[(a)] CDC/AAP case classification for moderate or severe periodontitis defined as ≥2 interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or ≥2interproximal sites with probing depth ≥5 mm, not on the same tooth

[(b)] Results are adjusted for year of NHANES survey

[(c)] Persons identifying racially as "Other" were omitted from this analysis due to the small number of these subjects (n=39)

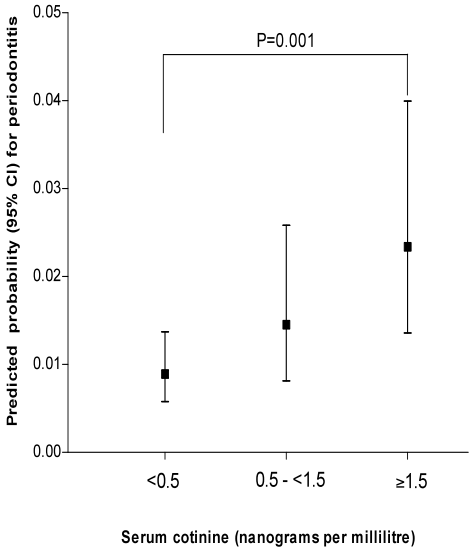

Figure 1: Predicted probability and 95% confidence interval (CI) of having moderate or severe periodontitis according to level of serum cotinine and adjusted for age, sex and year of NHANES, in dentate non-smoking United States (US) adults aged ≥20 years (n= 3137), NHANES 1999-2004*
*Periodontitis is defined using the CDC/AAP case classification for moderate or severe periodontitis: either ≥2 interproximal sites with clinical attachment level ≥4 mm, not on the same tooth, or ≥2interproximal sites with probing depth ≥5 mm, not on the same tooth

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